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Copper deficiency in ruminants

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Introduction

Copper deficiency, which causes metabolic problems in ruminants, is of serious concern to livestock producers because it can affect the livelihood of an entire region and cause farmers to suffer severe financial losses. Several decades of research have shown that its origins lie in the close relationships that exist among soils, plants, and animals.

Neonatal ataxia in sheep is a disease that originates in the soil and is associated with subnormal levels of copper in pastures and in the body of both ewes and affected lambs. The movements and gait of newborn and suckling lambs are uncoordinated because of degeneration of the central nervous system. In adult animals the fleece partly or completely loses its color and produces brittle, poor-quality wool. The sheep also suffer from anemia and diarrhea.

In cattle, copper deficiency retards growth and causes anemia, diarrhea, and loss of appetite. The coat loses its color and some animals die abruptly.

Factors that influence the disease

Outbreaks of this disease depend on several conditions. Geological and meteorological factors are very important.

Geological factors

Although the disease originates in the soil, it is not limited to a specific soil type but appears on a variety of soils. It is found on clayey, peaty, and sandy soils as well as ones that are near the ocean and therefore saline.

Climatic factors

A very hot summer followed by an autumn with little or late rainfall causes a considerable increase in the number of cases.

Influence of age

Although there is no set rule on the age of ewes that are likely to bear sick lambs, the disease seems to occur more frequently with older females. As animals age they form smaller copper reserves, and because of this decrease they may be more likely to give birth to diseased young. Although lambs absorb 71% of the copper they consume during the first weeks of life, the percentage falls gradually to 3–9% at adulthood. Thus, animals that suffer from deficiency at birth or in the following weeks will probably not have enough copper to meet their needs as adults. If they do reach adulthood, they are more likely than other animals to produce diseased offspring.

Influence of breed

The Merino sheep (wool breed) seems more susceptible than any of the meat breeds. The disorders caused by the disease mean severe financial losses for farmers, who must market wool of an inferior quality—if they can sell it at all.

Influence of the individual animal

Differences among individual animals are very pronounced. Some ewes give birth to ataxic lambs while others in the same flock produce perfectly normal offspring.

Symptoms

Sheep

In sheep, there are two forms of the disease, congenital neonatal ataxia or "swayback," and delayed neonatal ataxia.

In the first case, animals are born ataxic or become so in the first weeks of life. Symptoms vary greatly. Lambs may be stillborn or they may be completely paralyzed from birth and die within hours. Others show the same general problems with the nervous system (cerebral demyelination or cerebrospinal disorders) but appear only partly paralyzed or staggering; if their mothers are brought to them they can suckle almost normally. In severe cases of neonatal ataxia the animals may become anemic and even blind.

With delayed neonatal ataxia, lambs may be born diseased but their condition worsens only gradually during the first 3 weeks of life. They then die within a few days. If the disease appears a little later, however, at about 6 weeks of age, their chance of survival is greater. These animals can never recover completely: their hind limbs remain weak. Sometimes the bones are affected; fractures may occur before the nervous disorders appear. When the latter become evident the lambs walk on their fetlocks, their hind limbs become wobbly, and finally they fall down. The extreme effort the animal has to make to move even a few steps gives rise to a rapid heartbeat, or tachycardia.

In adult sheep, especially dark-colored animals, simple deficiency is easily diagnosed from the loss of color in their wool, which occurs in patches anywhere on their bodies. The wool becomes hard, brittle, very poor in quality, and greatly reduced in yield. Pregnant ewes suffering from copper deficiency have pale mucous membranes, a rapid pulse, sometimes a mild heart murmur, and obvious general signs of fatigue.

Overall, affected animals are in poor general condition characterized by progressive weight loss which may develop into anemia, doubtless caused by the almost uncontrollable diarrhea. As well as these main symptoms, adults may develop edemas (swellings) and bone problems which show up as extensive or severe malformations. Conjunctivitis and weeping eyes are also characteristic signs of bone disorders.

Cattle

In cattle, diagnosis of copper deficiency is based mainly on the violent, sickening diarrhea which degenerates into liquid stools of a grayish yellow color that may turn blackish. Their tails and hind limbs are very dirty and become tender from the frequent stools. Their bellies become thin, their mouths dry, and their tongues coated. The bodies of diseased animals give off a repulsive odor and their coats lose their color and become rough.

In black cows, the loss of color gives their coats a russet or very pale shade. This loss of pigment can also occur around the eyes, giving rise to the saying that the cows "are wearing spectacles."

The stiff gait later becomes an amble. This means that the animals move both right limbs at the same time, then both left limbs.

Some joints become swollen as well. The bones are fragile and young animals sometimes develop multiple fractures (of the humerus, femur, and ribs).

The disease progresses slowly, but occasionally animals may suddenly die of a heart attack. In these cases, cattle fall down and die abruptly after physical exertion. Some animals have problems with breathing.

The appetite of sick cattle is affected: they eat less grass or hay but continue to feed on concentrates. Milk production decreases because of the loss of appetite.

Anemia sometimes occurs, because copper plays an essential part in the release of iron stored in the liver.

The basic difference in symptoms between sheep and cattle is that diagnosis of the disease relies mainly on general disorders of the nervous system in sheep, and on diarrhea in cattle.

Lesions

Sheep

Both gross (visible) and microscopic lesions occur in sheep. In severe cases affecting very young lambs less than 3 weeks old, there are commonly gross lesions that appear as cavities and jelly-like areas in the white matter of the brain. Such lesions are rarer in lambs more than 3 weeks old and are never found in animals 6–8 months of age. Young lambs may develop symmetrical patches of softened white matter in the brain, accompanied by congestion or sometimes even edema, whereas older animals may show thickened areas in the fibrous tissue that sheaths the brain. The brain swells and its convolutions tend to disappear. In spite of this, the gray matter remains intact round the cavities caused by the disappearance of the white matter. In extreme cases of hydrocephaly (water on the brain), the volume of cerebrospinal fluid increases considerably and a large hernia of the cerebellum appears through the occipital opening.

In the case of microscopic lesions, there is a continual loss (demyelination) of substance from the membrane that covers the nerve

fibers. In the severest cases the covering membrane (myelin sheath) may disappear almost entirely from both hemispheres of the brain. The nuclei of nerve cells enlarge, then the vacuoles degenerate.

Cattle

Cattle develop bone deformations and heart lesions; spontaneous bone fractures occasionally occur in cattle grazing on copper-deficient herbages. Affected animals may show some bone deformations because cartilage grows normally but does not harden into bone. The outer walls of the bones become thinner, as do the columnar structures of porous bone material. The epiphyses, or ends of the long bones, become swollen.

The heart in diseased animals is generally much larger than in normal ones. Its arteries are surrounded by fat but it is flabby. The heart muscle lacks firmness and the internal walls of the ventricles are striped, spotted, or almost colorless. Reddish spots may appear on the surface of the myocardium.

Causes

Copper deficiency in sheep results from a defect in copper metabolism (or the absorption and use of copper in the body). In all countries of the world, copper levels in the blood and liver have always been abnormally low in afflicted animals. This deficiency is almost always present in females that have borne young. Its origin lies in the close ties that exist between soils and plants, which then affect the animals that pasture on the land.

Soils

Copper deficiencies occur on soils that very considerably. Soils that are very saline or too rich in molybdenum, although normal in copper content, may have the same effect as soils that contain too little copper (the copper level of the latter may be as low as 5.6 mg/kg of dry matter, whereas 8 mg/kg is normal).

Plants

Plants eaten by animals have varied copper contents. There are large differences from one country to another, but in copper-deficient plants the young shoots are yellowed and general growth is poor. Animals feeding on such plants become anemic, lose their appetite, and rapidly become very thin.

Simple and conditioned copper deficiency

Copper deficiency occurs in two forms, simple and conditioned. The simple kind occurs only in regions where copper levels in grass are less than normal (5–6 mg/kg of dry matter). In such conditions, ewes are highly likely to give birth to ataxic lambs. Conditioned copper deficiency

is found in areas where the copper content is normal (8 mg/kg of dry matter or more) but the metal cannot be absorbed and used normally because materials such as molybdenum or sulfates intervene and make it harder for animals to assimilate dietary copper. Indeed, molybdenum reacts with copper in the digestive tract of ruminants, forming complexes that block the biochemical activity of copper. Formation of the copper–molybdenum complex makes the copper impossible to use. If the ratio of copper to molybdenum in rations is close to 2, ruminants develop deficiency symptoms that resemble those of copper deficiency. In Canada, high levels of molybdenum have been reported in pastures in an area of Manitoba.

Sulfates reduce copper absorption, so rations rich in inorganic sulfates may cause ruminants to show symptoms exactly like copper deficiency even though their forage has a normal copper content and low molybdenum levels. If no molybdenum is present, mineral sulfates have no effect on copper metabolism. All this seems to show that there are metabolic interferences between the copper–molybdenum complex on the one hand and sulfates on the other, in the liver and in the blood serum.

Copper levels in animals

All these factors influence the copper level in animals. Copper is a micronutrient that is essential to animals, so particular attention has been paid to copper contents of the liver, the blood, the coat, and the milk of many healthy and diseased animals.

Copper content of the liver

Various studies have shown that the copper concentration in the liver can be used as an indicator of copper levels in an organism. In regions unaffected by copper deficiency, copper concentrations in the liver are more than 100 mg/kg of dry matter. In some cases the copper concentration may be as high as 400 mg/kg of dry matter.

Generally, the liver of young ruminants—sheep or cattle—contains more copper than that of adults. Below 20–30 mg/kg of dry matter, deficiency is a high risk and at concentrations lower than 10 mg/kg it is a certainty.

Copper content of the blood

The critical threshold level for copper in the blood is 0.6 µg/mL. In healthy animals levels are higher than 0.7 µg/mL. In ruminants with a severe deficiency, the copper level is around 0.1–0.3 µg/mL of blood.

Copper content of the coat

In the coat, the threshold value for copper is 8 mg/kg of dry matter. It may be as low as 3 mg/kg in deficient animals.

Copper content of the milk

Three major factors affect copper levels in milk: the breed, the stage of lactation, and the amount of copper the milk-producing animal can consume. Colostrum has a very high copper content, which later decreases in the milk. It is therefore extremely important that pregnant ewes receive a well-balanced diet. The copper level in a healthy ewe falls progressively from 0.20–0.60 mg/L of milk at the start of lactation to 0.04–0.16 mg/L a few months later.

Prevention and treatment

Sheep

It has been known for several decades that a lack of copper or excess of molybdenum and sulfur intake can cause simple or conditioned deficiencies in the animals. Livestock producers suffer heavy financial losses as a result. We tend to forget that the relationship between plants and animals is a basic element in the development of metabolic diseases. Healthy forage in fact has a copper content of 7–8 mg/kg of dry matter, and forage that is chlorotic, or straggling and growing poorly, contains no more than 2–3 mg/kg.

There are two ways of preventing this disease: the indirect method, by treating pastures, and the direct method, applied to the animals.

Copper reserves can be increased by applying copper sulfate to the soil at a rate of 5 kg/ha. Preferably the powder should be mixed well into the soil. The land should then produce good forage for 2–3 years. To avoid the risk of copper building up in sheep, which are very sensitive to it, be careful to apply the exact rate recommended and do not graze your flock on treated pasture for about 3 weeks. This time limit can be reduced if it rains heavily. Because of the close link between plants and animals, it is often sufficient (where possible) to transfer animals suffering from neonatal ataxia to grassland with a suitable copper content, for them to recover on their own.

However, if too much copper fertilizer is applied, the roots of the plants will benefit and not the aerial parts.

Foliar spraying has rarely been used so far on forages, probably because the technique is not yet well developed, it is necessary to spray again after each cut, and it is not easy to obtain solutions at the required concentrations.

This approach to the problem, that of treating the soil and plants, is effective mainly in the case of simple deficiencies. When conditioned deficiencies are present it is better to treat the animals.

Copper sulfate can be included in licking stones, in a proportion of 0.6–1%. This is a simple method, requiring practically no labor. However, it is difficult to supply the animals with the exact amount required because some use the stones more than others do. Copper sulfate can also be added to the drinking water of ruminants, but first you must make sure the containers are corrosion resistant.

Mineral supplements with high copper contents can be added to the rations of pregnant ewes, at a rate of 0.6% of copper sulfate. Such mineral supplements can be found on the market. However, all ewes do not tolerate them well, so you risk either causing chronic copper poisoning in the ewes or giving the newborn lambs too little protection.

Direct oral administration of copper is still practiced because the exact recommended dose can be given. Also, the farmer can check the state of health of his flock at the same time (for decalcification and parasitism, among other conditions), which is another benefit. However, it is difficult to treat a large flock by this method. It requires much time and effort to catch the animals one by one. Only 5% of the copper taken orally is absorbed through the wall of the intestines, and the sometimes rough handling of pregnant ewes carries the risk of abortions.

This oral method consists of giving pregnant ewes 20 mL of a 25% solution of copper sulfate as a single dose. Lambs less than 4 months old receive 5 mL, and those more than 4 months old are given 5–10 mL. To avoid the risk of poisoning, always dilute the copper sulfate with a large quantity of water for adults, or milk and water for lambs.

Copper can be administered parenterally. This method has much to recommend it because most of the dose administered collects in the liver, where the copper content increases rapidly and to a considerable degree.

Copper glycinate has replaced copper sulfate for this use, because the copper sulfate caused large lesions.

There are three ways of administering copper parenterally: by intramuscular injection, intravenous injection, or subcutaneous injection.

Intravenous injections, which can be given only by a qualified person, have gradually been replaced by intramuscular and subcutaneous injections. The last-named are best, especially for sheep and cattle, because they do not damage the carcass and there is no risk of cold abscesses, which can be caused by intravenous injections. As well, they leave only a small sore spot, the dose is absorbed slowly and has a longer-lasting effect, and they reduce the risk of poisoning.

A single subcutaneous injection of 25 mg of copper glycinate administered to ewes at about mid-pregnancy is generally sufficient to prevent ataxia in the newborn. The lambs should be given an injection at branding time, and then every 3 months.

Parenteral injections of copper compounds are definitely not advisable, however, for newborn lambs because they cause some deaths.

Cattle

In cattle, good results can be obtained from subcutaneous injection of 120 mg of copper glycinate, three times a year for animals less than a year old and twice a year for adults. Pregnant cows should be injected with 120 mg of copper glycinate every 3 months for best results.

Conclusions

It is clear, from studies of copper deficiency in ruminants, and especially sheep and cattle, that the disease is a serious one from both medical and economic points of view.

Because the soil is the prime factor in this disease, it is a wise precaution to obtain an analysis of the copper, molybdenum, and inorganic sulfate contents of your soil. Have your grass analyzed also.

If these tests reveal a deficiency, spread acid fertilizers on the soil before putting your animals out to pasture. Or else, provide them with copper supplements or inject them with copper glycinate.

Acknowledgments

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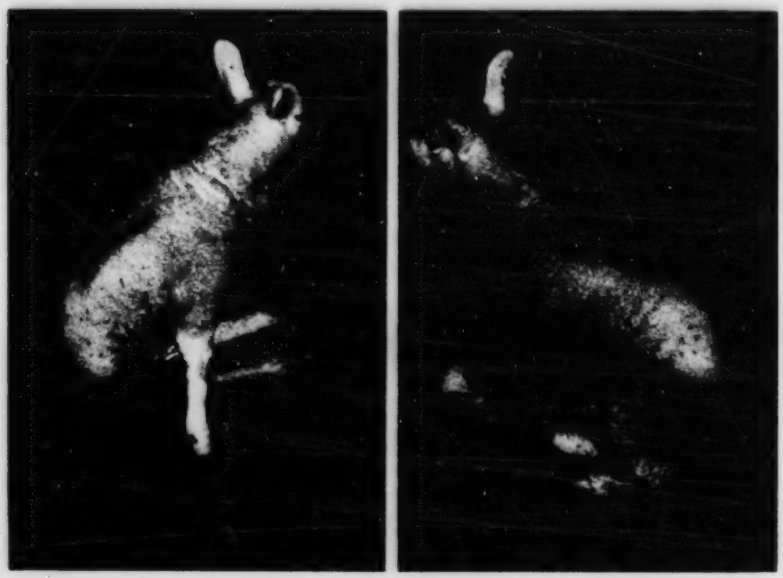
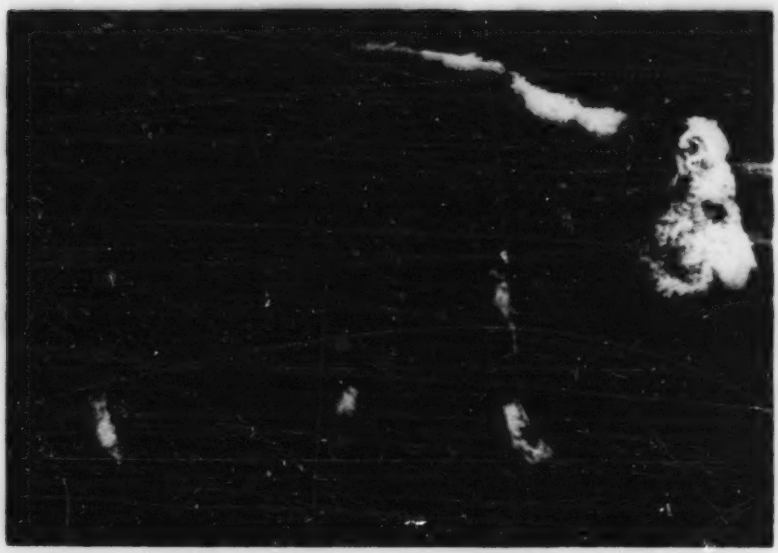


Lambs with ataxia caused by copper deficiency, showing problems with movement of hind limbs.

Heifer suffering from copper deficiency, showing a loss of color in its coat.

The same animal 3 months later, after treatment with copper glycinate.



Heifer with submaxillary edema induced by advanced copper deficiency.

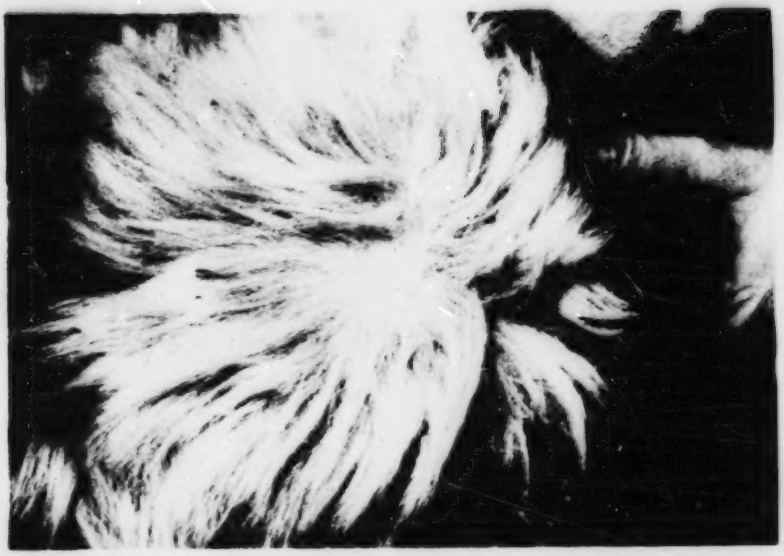
Animal suffering from diarrhea and with a tawny, pale coat.

Well-fleshed cow that has begun to scour.

Thin animal after loss of appetite.

Hereford cow on the left with a russet coat.

Animals with bristling, dull, pale coats.



Rough, bristling, grayish coat.

Calf with conditioned copper deficiency due to excess molybdenum; the animal shows severe growth retardation, expressed in the thickening of the long bones.

CONVERSION FACTORS

Metric units	Approximate conversion factors	Results in:
LINEAR		
millimetre (mm)	x 0.04	inch
centimetre (cm)	x 0.39	inch
metre (m)	x 3.28	feet
kilometre (km)	x 0.62	mile
AREA		
square centimetre (cm^2)	x 0.15	square inch
square metre (m^2)	x 1.2	square yard
square kilometre (km^2)	x 0.39	square mile
hectare (ha)	x 2.5	acres
VOLUME		
cubic centimetre (cm^3)	x 0.06	cubic inch
cubic metre (m^3)	x 35.31	cubic feet
	x 1.31	cubic yard
CAPACITY		
litre (L)	x 0.035	cubic feet
hectolitre (hL)	x 22	gallons
	x 2.5	bushels
WEIGHT		
gram (g)	x 0.04	oz avdp
kilogram (kg)	x 2.2	lb avdp
tonne (t)	x 1.1	short ton
AGRICULTURAL		
litres per hectare (L/ha)	x 0.089	gallons per acre
	x 0.357	quarts per acre
	x 0.71	pints per acre
millilitres per hectare (mL/ha)	x 0.014	fl. oz per acre
tonnes per hectare (t/ha)	x 0.45	tons per acre
kilograms per hectare (kg/ha)	x 0.89	lb per acre
grams per hectare (g/ha)	x 0.014	oz avdp per acre
plants per hectare (plants/ha)	x 0.405	plants per acre